02jul01 07:25:49 User208600 Session D1404.1
File 155:MEDLINE(R) 1966-2001/Jul W2 (c) format only 2001 Dialog Corporation

Set Items Description

Ref Items RT Index-term
E1 1 TETANUCLEATE
E2 1 TETANUKSEN
E3 13902 3 *TETANUS
E4 71 TETANUS --BLOOD --BL
E5 5 TETANUS --CEREBROSPINAL FLUID --CF
E6 30 TETANUS --CHEMICALLY INDUCED --CI
E7 12 TETANUS --CLASSIFICATION --CL
E8 393 TETANUS --COMPLICATIONS --CO
E9 21 TETANUS --CONGENITAL --CN
E10 338 TETANUS --DIAGNOSIS --DI
E11 3 TETANUS --DIET THERAPY --DH
E12 523 TETANUS --DRUG THERAPY --DT

Ref Items Type RT Index-term
R1 13902 3 *TETANUS
R2 4515 X DC=C1.252.410.90.217.864. (TETANUS)
R3 904 R 6 TRISMUS
R4 2533 B 7 CLOSTRIDIUM INFECTIONS

Ref Items Type RT Index-term
R1 2533 7 *CLOSTRIDIUM INFECTIONS
R2 2533 X DC=C1.252.410.90.217. (CLOSTRIDIUM INFECTIONS)
R3 122 B 9 BACILLACEAE INFECTIONS
R4 1785 N 10 BOTULISM
R5 3997 N 10 ENTEROCOLITIS, PSEUDOMEMBRANOUS
R6 279 N 3 ENTEROTOXEMIA
R7 1368 N 2 GAS GANGRENE
R8 13902 N 3 TETANUS

S1 13902 "TETANUS"
S2 2533 "CLOSTRIDIUM INFECTIONS"
S3 90754 THYROID
S4 38 S1 AND S3
S5 2 S2 AND S3

Ref Items RT Index-term
E1 753 THYROTROPIC
E2 2 THYROTROPICAL
E3 25841 8 *THYROTROPIN
E4 167 THYROTROPIN --ADMINISTRATION AND DOSAGE --AD
E5 30 THYROTROPIN --ADVERSE EFFECTS --AE
E6 17 THYROTROPIN --ANALOGS AND DERIVATIVES --AA
E7 896 THYROTROPIN --ANALYSIS --AN
E8 255 THYROTROPIN --ANTAGONISTS AND INHIBITORS --AI
E9 288 THYROTROPIN --BIOSYNTHESIS --BI
E10 10117 THYROTROPIN --BLOOD --BL
E11 12 THYROTROPIN --CEREBROSPINAL FLUID --CF
E12 6 THYROTROPIN --CHEMICAL SYNTHESIS --CS

S6 25841 "THYROTROPIN"
S7 19 S6 AND (S1 OR S2)
S8 30150 "THYROID GLAND"

Ref Items RT Index-term
E1 96 THYROID FUNCTION TESTS --VETERINARY --VE
E2 0 1 THYROID GALACTOSYLTRANSFERASE
E3 30150 5 *THYROID GLAND
E4 595 THYROID GLAND --ABNORMALITIES --AB
E5 1135 THYROID GLAND --ANALYSIS --AN
E6 992 THYROID GLAND --ANATOMY AND HISTOLOGY --AH
E7 672 THYROID GLAND --BLOOD SUPPLY --BS
E8 369 THYROID GLAND --CHEMISTRY --CH
E9 2573 THYROID GLAND --CYTOLOGY --CY
E10 1 THYROID GLAND --DIAGNOSIS --DI
E11 5066 THYROID GLAND --DRUG EFFECTS --DE
E12 601 THYROID GLAND --EMBRYOLOGY --EM

Ref Items Type RT Index-term
R1 30150 5 *THYROID GLAND
R2 30150 X DC=A6.407.900. (THYROID GLAND)
R3 221 R 3 STRUMA OVARII
R4 11014 R 3 THYROIDECTOMY
R5 217 R 3 ULTIMOBRANCHIAL BODY
R6 5409 B 30 ENDOCRINE GLANDS

S9 17616 CLOSTRID?
S10 7 S8 AND S9
S11 133 PHOSPHOLIPASE AND NEUROTOXIN
S12 27833 THYROX?
S13 2 S9 AND S12
S14 6426 BOTULI?
S15 0 S14 AND S12
S16 0 S 14 AND S8
S17 18 S3 AND S14

4/6/1 11245935 21195143 PMID: 11298123
Intradermal skin test with diabetes specific antigens in patients with type 1 diabetes. Mar 2001

4/6/2 10726207 20373526 PMID: 10916814
[Preventive activities in primary health care: identifying the agreement among evidence-based guidelines] Actividades preventivas en atencion primaria: identificacion de areas de concordancia entre guias de practica clinica basadas en la evidencia. 2000

4/6/3 10722524 20424529 PMID: 10970112
Muscle reinnervation in hypothyroid rats. 1996

4/6/4 10042358 99134381 PMID: 9933650
Detection of novel carbohydrate binding activity of interleukin-1. Feb 12 1999

4/6/5 09732112 98204670 PMID: 9545109
Induction of oral tolerance in human autoimmune thyroid disease. Mar 1998

4/6/6 09716948 98187354 PMID: 9526606
Preliminary studies with recombinant chorionic gonadotropin beta-subunit produced in Escherichia coli for use as an antigen in a birth control vaccine. Mar 1998

4/6/7 09511341 95156316 PMID: 7853237
Effects of thyroid hormone on fast- and slow-twitch skeletal muscles in young and old rats. Nov 15 1994

4/6/8 09419224 97479154 PMID: 9337806
Midlife periodic health exam in the primary care practice. Oct 1997

4/6/9 09175752 96429836 PMID: 8832986
Elevated serum prolactin or elevated prolactin/cortisol ratio are associated with autoimmune processes in systemic lupus erythematosus and other connective tissue diseases. Mar 1996

4/6/10 09073399 97025581 PMID: 8871760
The pathogenicity of spontaneously-occurring thyroglobulin-reactive T lymphocytes from BB/WOR rats. 1996

4/6/11 08491947 95229821 PMID: 7714099
Activation of T lymphocyte subsets by synthetic TSH receptor peptides and recombinant glutamate decarboxylase in autoimmune thyroid disease and insulin-dependent diabetes. Apr 1995

4/6/12 08365713 95213731 PMID: 7699384
Response of fast muscle innervation to hypothyroidism. Dec 1 1994

4/6/13 07938907 93390517 PMID: 8377760
[Evaluation on our procedure for autotransplantation of parathyroid glands by the intact-PTH] Aug 1993

4/6/14 07651909 93027039 PMID: 1408659
The time course of thyroid-hormone-induced changes in the isotonic and isometric properties of rat soleus muscle. Jul 1992

4/6/15 07355645 90368987 PMID: 2168443
Thyroid-stimulating antibody activity between different immunoglobulin G subclasses. Sep 1990

4/6/16 07344123 90192059 PMID: 2138281
The rate of tetanic relaxation is correlated with the density of calcium ATPase in the terminal cisternae of thyrotoxic skeletal muscle. Jan 1990

4/6/17 06343059 88056008 PMID: 3678672
Screening practices of family physicians: a comparison of STFM and AAFP members. Sep-Oct 1987

4/6/18 06175404 85196529 PMID: 3887952
Characteristics and bioefficacy of monoclonal antigonadotropin releasing hormone antibody. Mar 1985

4/6/19 05788755 89067487 PMID: 2848890
Probing the normal and autoimmune B cell repertoire with Epstein-Barr virus. Frequency of B cells producing monoreactive high affinity autoantibodies in patients with Hashimoto's disease and systemic lupus erythematosus. Dec 15 1988

4/6/20 05639197 88043483 PMID: 3118511
Temporal analysis of dithiobiuret neurotoxicity in rats and assessment of potential nonneural causes. Nov 1987

4/6/21 05514295 86248155 PMID: 2424791
Enhancement of antigonadotropin response to the beta-subunit of ovine luteinizing hormone by carrier conjugation and combination with the beta-subunit of human chorionic gonadotropin. Jul 1986

4/6/22 05330203 90033436 PMID: 2806615
Antibody response and characteristics of antibodies in women immunized with three contraceptive vaccines inducing antibodies against human chorionic gonadotropin. Nov 1989

4/6/23 05287508 89332330 PMID: 2756340
Affinity purification of IgG subclasses and the distribution of thyroid auto-antibody reactivity in Hashimoto's thyroiditis. Jul 1989

4/6/24 05235053 88154038 PMID: 3257970
Microsomal antigen-reactive lymphocyte lines and clones derived from thyroid tissue of patients with Graves' disease. Apr 1988

4/6/25 04893804 80227723 PMID: 7391005
Interaction of fragments B and C of tetanus toxin with neural and thyroid membranes and with gangliosides. Jul 10 1980

4/6/26 04805868 82247718 PMID: 6179073
[Possible relation between the immune response and thymus-dependence of the immunizing antigens in children in an endemic goiter region] O vozmozhnoi sviazi mezhdu immunnym otvetom i timuszavisimost'iu immuniziruiushchikh antigenov u detei v ochage zobnoi endemii. May-Jun 1982

4/6/27 04760092 84253338 PMID: 6331133
Gangliosides, the thyrotropin receptor, and autoimmune thyroid disease. 1984

4/6/28 04713242 80150428 PMID: 6244725
Thyrotropin receptors and gangliosides. 1980

4/6/29 04661467 84263155 PMID: 6378767
Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease. Jul 1984

4/6/30 04655785 84158405 PMID: 6368201
Characterization of monoclonal antibody 3G5 and utilization of this antibody to immobilize pancreatic islet cell gangliosides in a solid phase radioassay. Apr 1984

4/6/31 03639753 82119926 PMID: 7328074
Thyroid function in tetanus. Jul 1981

4/6/32 03528268 79020956 PMID: 212049
Structure:function studies of receptors for thyrotropin and tetanus toxin: lipid modulation of effector binding to the glycoprotein receptor component. Jul 14 1978

4/6/33 03392187 79173087 PMID: 220221
Tetanus toxin and thyrotropin interactions with rat brain membrane preparations. May 25 1979

4/6/34 03389313 79127176 PMID: 217603

Tetanus toxin interactions with the thyroid: decreased toxin binding to membranes from a thyroid tumor with a thyrotropin receptor defect and in vivo stimulation of thyroid function. Mar 1978

4/6/35 03345587 76185875 PMID: 131619
[Permeability of sublingual mucosa to organic molecules. Limited role of sublingual absorption in aerosol vaccinations] La permeabilite de la [redacted]use sub-linguale aux molecules organiques. Les limites du role de [redacted] sub-linguale dans la vaccination par aerosols 1975

4/6/36 03008262 76102749 PMID: 813223
Isoimmunization against human chorionic gonadotropin with conjugates of processed beta-subunit of the hormone and tetanus toxoid. Jan 1976

4/6/37 02679440 77187913 PMID: 193853
[redacted]s toxin interactions with thyroid plasma membranes. In[redacted]ns for structure and function of tetanus toxin receptors and potential pathophysiological significance. Jun 25 1977

4/6/38 01235275 70276257 PMID: 4916539
Immunosuppressive therapy for the eye changes of Graves' disease. Sep 1970

4/7/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
10726207 20373526 PMID: 10916814
[Preventive activities in primary health care: identifying the agreement among evidence-based guidelines] Actividades preventivas en atencion primaria: identificacion de areas de concordancia entre guias de practica clinica basadas en la evidencia.
Gosalbes Soler V; Marquez Calderon S; Maiques Galan A; Latour Perez J; Bernal Delgado E; Puig Barbera J; Arranz Lazaro M
Unidad Docente de Medicina Familiar y Comunitaria, Valencia. gosalbesvic@gua.es
Medicina clinica (SPAIN) 2000, 114 Suppl 2 p88-92, ISSN 0025-7753 Journal Code: LTQ
Languages: SPANISH Document type: Journal Article Record type: Completed
BACKGROUND: The purpose of this article is to identify the agreement among evidence-based guidelines about recommendations on preventive activities in low risk adults. METHODS: For which we identified, from the 1996 US Task Force on Preventive Services Guide those preventives activities classified like A or B (recommendation in favour of provision) and like D or E (recommendation against provision), excepting those D and E recommendations based on descriptives studies or expert opinions. Both preventive activities aimed at pregnant women and children and those which are not applicable to our context were excluded. Selected preventive services were compared with the range of age, sex and periodicity in which agreement with the recommendations of American College of Physicians and Canadian Task Force on Preventive Services existed. RESULTS: We found the following agreements. Screening activities. In favour: screening for hypercholesterolemia, hypertension, breast cancer, colorectal cancer, uterine cervix cancer, rubella, visual and hearing impairment and problem drinking. Against: cancer of prostate, lung, bladder and thyroid , and asymptomatic bacteriuria. Counseling activities. In favour: smoking, motor-vehicles injuries, alcohol consumption, unintended pregnancy. Immunizations and quimioprophylaxis. In favour: Vaccines for influenza, tetanus -diphtheria, hepatitis B and measles-mumps-rubella. Postexposure prophylaxis to hepatitis A, hepatitis B, meningococcal, rabies and tetanus . CONCLUSIONS: We see then, that a high degree in agreement among the main guidelines exists; about the preventive activities to perform in Primary Health Services, nevertheless we observed low fulfillment of certain preventive activities in Primary Health Services, different barriers for the accomplishment from these activities were described. Record Date Created: 20000919

4/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
09732112 98204670 PMID: 9545109
Induction of oral tolerance in human autoimmune thyroid disease.
Lee S; Scherberg N; DeGroot LJ
Department of Medicine, The University of Chicago, Illinois, USA.
Thyroid (UNITED STATES) Mar 1998, 8 (3) p229-34, ISSN 1050-7256 Journal Code: BJW
Languages: ENGLISH Document type: Clinical Trial; Journal Article; Randomized Controlled Trial Record type: Completed
Our laboratory has reported suppression of experimental autoimmune thyroiditis in mice by oral feeding with antigen. Based on these data, we considered it possible that oral feeding of animal thyroglobulin (TG) might induce tolerance to antigen in human autoimmune thyroid disease (AITD). Thirteen patients receiving thyroid hormone replacement with synthetic thyroxine (T4) (five patients with Graves' disease, treated with radioiodine 4 to 11 years ago and eight patients with Hashimoto's thyroiditis) were randomly assigned to a test group (switched to replacement with desiccated thyroid from porcine thyroids) and a control group (maintained on synthetic T4). Humoral and cellular immunologic parameters were evaluated in addition to clinical parameters before and every 3 months after the onset of study for a year. At the onset of study, there was no difference in clinical parameters, or humoral and cellular immunity to thyroid autoantigens, except a finding that one thyroid peroxidase (TPO) peptide (100 approximately 119) appeared to stimulate peripheral blood mononuclear cells (PBMC) during in vitro microproliferation assay more in the test group than control group ($p = 0.051$ by t test). Additionally, almost all of TPO and thyrotropin receptor extracellular domain (TSHR) peptides were slightly more stimulatory to PBMC from the test group than the control group, although this was not statistically significant. After treatment, all variables were analyzed at each time point between groups (t test), and also were analyzed over time in each group (analysis of variance, ANOVA). Among the clinical parameters, thyrotropin (TSH) levels were unchanged and equal. Total serum T4 levels ($p < 0.05$ at 6 and 12 months after treatment) and free thyroxine indices (FT4I) ($p < 0.05$ at all time points after treatment) were lower in the test group than the control group. This is an expected result of treatment with desiccated thyroid . We found no change over time nor any difference between groups at time points for titers of antibodies to thyroid autoantigens, ie, human TG, human TPO, and recombinant human TSHR from Escherichia coli. However, cellular immunity, measured by in vitro microproliferation of PBMC to peptides of TPO or TSHR, showed significant differences between groups. At 12 months, stimulatory indices (SI) of PBMC to six peptides, containing the indicated amino acids (764 approximately 95, 100 approximately 119, 110 approximately 129, 261 approximately 275, 441 approximately 448, 708 approximately 727) of 10 TPO peptides, and one peptide (145 approximately 163) of 14 TSHR peptides were lower in the test group than control group ($p < 0.05$). SI of PBMC to phytohemagglutinin, purified protein derivative from mycobacteria, and tetanus toxoid were not different between groups nor changed over time in any group. In conclusion, treating patients with AITD with an antigen related to the autoantigen TG did not produce changes in humoral immunity parameters, while cellular immunity to certain peptides were apparently suppressed. While the results are both surprising and intriguing, we need more evidence to justify the use of autoantigen as a form of immunospecific therapy in patients with AITD. Record Date Created: 19980515

4/7/7 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
09511341 95156316 PMID: 7853237
Effects of thyroid hormone on fast- and slow-twitch skeletal muscles in young and old rats.
Larsson L; Li X; Teresi A; Salviati G
Department of Clinical Neurophysiology, Karolinska Hospital, Stockholm, Sweden.
Journal of physiology (ENGLAND) Nov 15 1994, 481 (Pt 1) p149-61, ISSN 0022-3751 Journal Code: JQV Languages: ENGLISH Document type: Journal Article Record type: Completed
1. The effects of 4 weeks of thyroid hormone treatment on contractile, enzyme-histochemical and morphometric properties and on the myosin isoform composition were compared in the slow-twitch soleus and the fast-twitch extensor digitorum longus (EDL) muscle in young (3-6 months) and old (20-24 months) male rats. 2. In the soleus of untreated controls, contraction and half-relaxation times of the isometric twitch increased by 19-32% with age. The change in contractile properties was paralleled by an age-related increase in the proportions of type I fibres and type I myosin heavy chain (MHC) and slow myosin light chain (MLC) isoforms. 3. In the EDL of controls, contraction and half-relaxation times were significantly prolonged (21-38%) in the post- tetanus twitch in the old animals. No significant age-related changes were observed in enzyme-histochemical fibre-type proportions, although the number of fibres expressing both type IIA and IIB MHCs and of fibres expressing slow MLC isoforms was increased in the old animals. 4. Serum 3,5,3',5'-tetraiodothyronine (T4) levels were lower (34%) in the old animals, but the primary byproduct of T4, 3,5,3'-triiodothyronine (T3), did not differ between young and old animals. 5. The effects of 4 weeks of thyroid hormone treatment were highly muscle specific, and were more pronounced in soleus than in EDL, irrespective of animal age. In the soleus, this treatment shortened the contraction and half-relaxation times by 35-57% and decreased the number of type I fibres by 66-77% in both young and old animals. In EDL, thyroid hormone treatment significantly shortened the contraction time by 24%, but the change was restricted to the old animals. 6. In conclusion, the ability of skeletal muscle to respond to thyroid hormone treatment was not impaired in old age and the age-related changes in speed of contraction and enzyme-histochemical properties and myosin isoform compositions were diminished after thyroid hormone treatment in both the soleus and EDL. Record Date Created: 19950314

4/7/12 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
08365713 95213731 PMID: 7699384
Response of fast muscle innervation to hypothyroidism.
Cuppini R; Sartini S; Ambrogini P; Gallo G
Istituto di Anatomia e Fisiologia, Universita di Urbino, Italy.
Journal of the neurological sciences (NETHERLANDS) Dec 1 1994, 127 (1) p107-13, ISSN 0022-510X Journal Code: JBJ Languages: ENGLISH Document type: Journal Article Record type: Completed
The early period of motor innervation development is characterized by multiple innervation of muscle cells. This transitory state in rat extensor digitorum longus (edl) muscle is normally concluded at weaning when a 1:1 ratio between nerve endings and muscle cells is reached. Motor innervation of edl muscle in rats made hypothyroid after weaning was studied in three ways: electrophysiology (intracellular recordings of muscle postsynaptic potentials) was carried out to study neuromuscular transmission; silver impregnation of terminal axons to observe sprouting; force production in twitch and tetanus following direct muscle stimulation and nerve stimulation. A number of multiply innervated muscle cells was found in hypothyroid rats following two months of treatment. This finding seems to be related to the appearance of nodal sprouting in motor axons. No sign of denervated end-plates was found. Twitch and tetanus tension were smaller than in controls, but they were bigger when referred to unitary muscle mass. Time course of twitch, particularly half relaxation, was slowed in muscles of hypothyroid rats. These findings suggest that plastic processes occur in muscle innervation of rats made hypothyroid after weaning. Therefore, thyroid hormones play a role in stabilizing motor innervation not only during development, but also in adults. Record Date Created: 19950428

4/7/13 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
07938907 93390517 PMID: 8377760
[Evaluation on our procedure for autotransplantation of parathyroid glands by the intact-PTH]
Ueda M; Funahashi H; Sato Y; Kato M; Takagi H
Second Department of Surgery, Nagoya University, School of Medicine, Japan.
Nippon Geka Gakkai zasshi (JAPAN) Aug 1993, 94 (8) p840-6, ISSN 0301-4894 Journal Code: NGG Languages: JAPANESE Document type: Journal Article Record type: Completed
In thyroid cancer surgery since 1978 we have made parathyroidal autotransplantation which has been to resect the entire parathyroid, cut fine to mud and autotransplant into greater pectoral muscle. To evaluate our procedure and consider the mechanism of hypocalcemia and tetanus following surgery, we examined the intact-PTH recovered to about 80% of its preoperative value on 14-postoperative-days and thereafter remained almost constant. It remained below the sensitivity of measurement from immediately to 3-postoperative-days, and with our procedure, parathyroid was considered to be totally resected. Comparison between 2 (6 cases) and more autotransplanted glands (11 cases) revealed that the former showed slightly later functional recovery, but recovered to the almost some extent as the other 21-postoperative-days. Comparison between the group requiring calcium supplement therapy (12 cases) and the otherwise (5 cases) revealed almost the same course of recovery. Thus, our procedure seems to enable us to make satisfactory functional preservation and expect functional recovery by 2-gland

autotransplantation at least. The supplement therapy was considered to be detrimental to the take of autotransplanted glands and functional recovery, and no correlation was noted between the onset of tetanus and intact-PTH. Record Date Created: 19931020

4/7/17 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
06343059 88056008 PMID: 3678672
Screening practices of family physicians: a comparison of STFM and AAFP members.
Resnicow K; Schorow M; Bloom HG; Massad R; Coll-Barth M
American Health Foundation, New York City.
Family medicine (UNITED STATES) Sep-Oct 1987, 19 (5) p341-5, ISSN 0742-3225 Journal Code: FAL Languages: ENGLISH Document type: Journal Article Record type: Completed
The screening practices of 146 members of the Society of Teachers of Family Medicine (STFM) and 129 members of the American Academy of Family Physicians (AAFP) are compared. The screening practices of physicians from the two organizations were generally similar for psychosocial and behavioral problems, many forms of cancer, and numerous other conditions considered for inclusion in the routine periodic screening of asymptomatic individuals. However, for numerous diseases and tests, the screening practices of physicians from the two groups were significantly different. AAFP physicians were more likely to screen for lung and skin cancer, thyroid dysfunction, diabetes, and anemia, AAFP physicians were more likely to utilize chest x-ray, ECG, urinalysis, and SMA 6/12. STFM physicians were more likely to perform gonoccocal culture and tetanus -diphtheria immunization as well as to inquire about seat belt use. Three variables were found to predict physician screening practices as well as to account for the differences found between physicians drawn from the two organizations: completion of a residency in family medicine, year of graduation from medical school, and number of patients seen per week. Physicians reported practices were compared with recommendations in the major critical reviews: Frame and Carlson, Breslow and Sommers, the Canadian Task Force, and the American Cancer Society. For a number of tests and diseases physicians' reported practices were divergent with recent recommendations. Record Date Created: 19871221

4/7/20 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05639197 88043483 PMID: 3118511
Temporal analysis of dithiobiuret neurotoxicity in rats and assessment of potential nonneural causes.
Williams KD; Miller MS; Boysen BG; Peterson RE
School of Pharmacy, University of Wisconsin, Madison 53706.
Toxicology and applied pharmacology (UNITED STATES) Nov 1987, 91 (2) p212-21, ISSN 0041-008X Journal Code: VWO Contract/Grant No.: 1-K04-ES00098, ES, NIEHS; ES01906, ES, NIEHS Languages: ENGLISH Document type: Journal Article Record type: Completed
To evaluate the hypothesis that depressed neuromuscular transmission causes dithiobiuret (DTB)-induced muscle weakness in rats, the temporal development of impaired treadmill performance and deficits in the nerve-elicited muscle contractions were compared during daily treatment with the toxicant (DTB, 1 mg/kg/day X 6 days). Diminished treadmill test performance after 4 days of treatment marked the initial detection of impaired motor function. At this time fading (loss of tension during tetanus) of gastrocnemius contractions elicited in response to 100-Hz sciatic nerve stimulation occurred in DTB-treated rats but not in controls. After 5 and 6 days of treatment, treadmill failure became complete, tetanic fade worsened dramatically, and peak contractile tension measured during trains of nerve stimulation (10-250 Hz) decreased progressively. Appearing by Day 6 were marked body weight loss, dehydration, hypothermia, and a depression in serum concentrations of thyroid hormones. Total oxygen content of the blood was not reduced at any time during treatment, and serum concentrations of glucose, sodium, potassium, calcium, chloride, and phosphorus in DTB-treated rats on Day 6 were similar to those of control animals. Therefore, hypoxia, hypoglycemia, or a serum electrolyte imbalance do not initiate or modulate the neuromuscular toxicity. Light microscopic evaluation of liver, kidney, lung, thyroid , and other organs in intoxicated rats was unremarkable and in skeletal muscles and selected sites of brain, spinal cord, and sciatic nerve no morphologically significant lesions were observed. Even when DTB-intoxicated rats were maintained in a state of flaccid muscle weakness for 5 continuous days, peripheral nerve lesions proximal to the intramuscular nerves were not detected. Thus, depressed neuromuscular transmission appears to be the primary cause of the flaccid muscle weakness and no evidence was obtained that nonneural effects of DTB initiate or modulate this effect. Record Date Created: 19871211

4/7/24 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05235053 88154038 PMID: 3257970
Microsomal antigen-reactive lymphocyte lines and clones derived from thyroid tissue of patients with Graves' disease.
Fisfalen ME; DeGroot LJ; Quintans J; Franklin WA; Soltani K
Department of Medicine, University of Chicago, Illinois 60637.
Journal of clinical endocrinology and metabolism (UNITED STATES) Apr 1988, 66 (4) p776-84, ISSN 0021-972X Journal Code: HRB Contract/Grant No.: AM-13377, AM, NIADDK; CA19589, CA, NCI; DK-27384, DK, NIDDK
Languages: ENGLISH Document type: Journal Article Record type: Completed
Thyroid mononuclear cells (TMC) were maintained in long term cocultures with thyroid fibroblasts and thyroid epithelial cells from patients with Graves' disease, using medium supplemented with thyroid microsomal antigen (McAg) and IL-2. The TMC consisted predominantly of T4+ (CD4+, helper) and, to a lesser extent, T8+ (CD8+, cytotoxic/suppressor) lymphocytes, with a small number of macrophages and natural killer cells. The average T4+ to T8+ ratio was 3.2. From these cultures we obtained thyroid T cell lines and clones reactive to thyroid antigens. T Cell lines were tested in a microproliferation assay using thyroglobulin (Tg), McAg, tetanus toxoid, and IL-2. Of 14 lines from 6 patients, 2 proliferated in response to McAg when TMC plus thyroid fibroblasts were used as antigen-presenting cells. Clones of thyroid lymphocytes were obtained by culturing cells at limiting dilution with IL-2, McAg, and different types of autologous accessory cells. Peripheral blood mononuclear cells and skin fibroblasts provided the best source of accessory cells, allowing near 100% cloning efficiency. Of 26 clones tested, 6 recognized McAg, 2 were Tg reactive, and 3 were autoreactive. All phenotyped clones were of the T4+ phenotype. Our method results in production of thyroid T cell lines and clones. The fibroblasts probably provided growth factors and/or collaborated with peripheral blood mononuclear cells as antigen-presenting cells. These lines and clones from patients with Graves' disease were predominantly helper T cells, in contrast to the previously demonstrated cytotoxic/suppressor cell predominance in cells from patients with Hashimoto's thyroiditis. This difference in cell function may help explain the differing clinical courses of these two closely related autoimmune thyroid diseases. The availability of long term microsomal antigen-specific T cell clones should allow careful analysis of the role these cells play in thyroid autoimmunity. Record Date Created: 19880419

4/7/25 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
04893804 80227723 PMID: 7391005
Interaction of fragments B and C of tetanus toxin with neural and thyroid membranes and with gangliosides.
Morris NP; Consiglio E; Kohn LD; Habig WH; Hardegree MC; Helting TB
Journal of biological chemistry (UNITED STATES) Jul 10 1980, 255 (13) p6071-6, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19800928

4/7/27 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
04760092 84253338 PMID: 6331133
Gangliosides, the thyrotropin receptor, and autoimmune thyroid disease.
Lacetti P; Tombaccini D; Aloj S; Grollman EF; Kohn LD
Advances in experimental medicine and biology (UNITED STATES) 1984, 174 p355-67, ISSN 0065-2598 Journal Code: 2LU Languages: ENGLISH Document type: Journal Article Record type: Completed
The thyrotropin (TSH) receptor has been proposed to be composed of a membrane glycoprotein and a membrane ganglioside, the former important in high affinity recognition, the latter vital for message coupling to the adenylate cyclase system. The present study used two approaches, formation of antireceptor monoclonal antibodies and reconstitution, to validate the model and further examine the role of the ganglioside. Three kinds of monoclonal antireceptor antibodies are defined. One group which inhibits TSH binding and TSH functions, i.e., TSH-stimulated adenylate cyclase activity, iodide uptake, and thyroid hormone release, is shown to be directed against the glycoprotein component of the receptor. The second group includes antibodies which mimic TSH in all stimulatory actions, are competitive agonists of TSH, are equivalent to thyroid stimulating antibodies in the sera of patients with Graves' disease, and are directed against the ganglioside component of the receptor. These stimulating monoclonal antibodies are directed against a minor ganglioside membrane component which fractionates as a disialoganglioside. When this ganglioside is incorporated into 1-8 thyroid cells which have a correlated ganglioside deficiency and TSH receptor defect, reconstitution of TSH stimulated adenylate cyclase activity occurs. Whereas the first group of antibodies inhibits TSH-stimulated function, they do not inhibit the stimulatory antibodies which mimic TSH, an observation consistent with the 2 component hypothesis of the receptor model. The third group of antibodies have a mix of properties from the first two groups and suggests that the TSH receptor in situ is an actual complex of the two components or that there are common carbohydrate determinants in the functional sites of each receptor component. Implications of a TSH receptor structure in which its ganglioside and glycoprotein components are in equilibrium with pools of free components and, in turn, components important for cholera toxin, tetanus toxin and interferon receptors are discussed. In regard to the pathogenesis of Graves' disease, the data indicate that thyroid stimulating autoantibodies are autoimmune equivalents of cholera toxin with respect to the importance of ganglioside function. Since antiidiotype studies of antibodies against TSH confirm a structural relationship between receptors for thyrotropin, cholera toxin, and thyroid stimulating autoantibodies, the data establish an unequivocal role for the ganglioside in TSH receptor structure which facilitates interpretation of in vitro experiments aimed at understanding the mechanism of ganglioside-ligand interactions. Record Date Created: 19840823

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04713242 80150428 PMID: 6244725
Thyrotropin receptors and gangliosides.
Kohn LD; Consiglio E; DeWolf MJ; Grollman EF; Ledley FD; Lee G; Morris NP
Advances in experimental medicine and biology (UNITED STATES) 1980, 125 p487-503, ISSN 0065-2598 Journal Code: 2LU Languages: ENGLISH Document type: Journal Article; Review Record type: Completed (44 Refs.) Record Date Created: 19800530

4/7/29 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
04661467 84263155 PMID: 6378767
Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease.
Fawcett J; Hutton C; Mclachlan SM; Clark F; Rees Smith B
Immunology (ENGLAND) Jul 1984, 52 (3) p525-8, ISSN 0019-2805 Journal Code: GH7
Languages: ENGLISH Document type: Journal Article Record type: Completed
The humoral immune response to tetanus toxoid has been studied in patients with Hashimoto's disease. Although the magnitude of the response was similar to that observed in normal subjects, the Hashimoto patients demonstrated an inability to regulate their levels of tetanus toxoid antibody. This apparent defect in the control of antibody synthesis may be an important factor in both the initiation and perpetuation of autoimmune thyroid disease. Record Date Created: 19840829

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03639753 82119926 PMID: 7328074

Thyroid function in tetanus.
Dastur FD; Shah J; Awatramani V; Shah JM; Pardiwalla BS; Nair KG; Mehta MN; Desai KB
Journal of the Association of Physicians of India (INDIA) Jul 1981, 29 (7) p519-25, ISSN 0004-5772 Journal Code: HG7 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19820412

4/5/34 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
03389313 79127176 PMID: 217603
Tetanus toxin interactions with the thyroid: decreased toxin binding to membranes from a thyroid tumor with a thyrotropin receptor defect and in vivo stimulation of thyroid function.
Habig WH; Grollman EF; Ledley FD; Meldolesi MF; Aloj SM; Hardegree MC; Kohn LD
Endocrinology (UNITED STATES) Mar 1978, 102 (3) p844-51, ISSN 0013-7227 Journal Code: EGZ Languages: ENGLISH Document type: Journal Article Record type: Completed
Normal rat thyroid membranes adsorb neurotoxicity when incubated with purified tetanus toxin. Membranes from a rat thyroid tumor with a thyrotropin receptor defect adsorb very little neurotoxicity when similarly evaluated. This inability of the tumor membranes to adsorb neurotoxicity is correlated with a defect in their ability to bind both 125I-labeled tetanus toxin and [125I]iodothyrotropin. The effect of tetanus toxin on the release of radioiodine from the thyroids of appropriately prepared mice has been measured by adapting methods used for the bioassay of thyrotropin. One minimum lethal dose of tetanus toxin given sc caused a significant release of radioiodine into the blood of mice 48 h after injection. In mice subjected to the stress of prior bleedings or anesthesia, the release of radioiodine from the thyroid by tetanus toxin was accelerated, i.e., the increase in blood radioiodine could be measured 24 h after injection. These results again suggest that tetanus toxin may interact with thyrotropin receptors on thyroid plasma membranes. The "sympathetic overactivity syndrome" seen in some patients with tetanus and the syndrome characterized as " thyroid [illegible]" in patients with Graves' disease are discussed as they may relate to these observations. [illegible]d Date Created: 19790516
Tags: Animal
Descriptors: Protirelin--metabolism--ME; *Receptors, Cell Surface --metabolism--ME; * Tetanus Toxin--metabolism--ME; * Thyroid Gland --metabolism--ME; * Thyroid Neoplasms--metabolism--ME; Cell Membrane--metabolism--ME; Kinetics; Rats
CAS Registry No.: 0 (Receptors, Cell Surface); 0 (Tetanus Toxin);24305-27-9 (Protirelin)

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[Various findings on the processes of natural detoxication of the body during infectious processes] Nekotorye dannye o protsessakh estestvennoi detoksikatsii organizma pri infektsionnykh protsessakh. Oct 1967

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Clostridium septicum infection of the thyroid gland.
Warren CP; Mason BJ
Postgraduate medical journal (ENGLAND) Sep 1970, 46 (539) p586-8, ISSN 0032-5473 Journal Code: PFX
Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19710204
Tags: Human; Male
Descriptors: Clostridium Infections --complications--CO; *Thyroiditis-etiology--ET; Adenocarcinoma--complications--CO; Clostridium--isolationand purification--IP; Colonic Neoplasms--complications--CO; Middle Age;Thyroiditis--microbiology--MI

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Gangliosides, the thyrotropin receptor, and autoimmune thyroid disease. 1984

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Thyroid function in tetanus. Jul 1981

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Tetanus toxin and thyrotropin interactions with rat brain membrane preparations. May 25 1979

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Tetanus toxin interactions with the thyroid: decreased toxin binding to membranes from a thyroid tumor with a thyrotropin receptor defect and in vivo stimulation of thyroid function. Mar 1978

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03639753 82119926 PMID: 7328074
Thyroid function in tetanus.
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Habig WH; Grollman EF; Ledley FD; Meldolesi MF; Aloj SM; Hardegree MC; Kohn LD
Endocrinology (UNITED STATES) Mar 1978, 102 (3) p844-51, ISSN 0013-7227 Journal Code: EGZ Languages: ENGLISH Document type: Journal Article Record type: Completed
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CAS Registry No.: 0 (Receptors, Cell Surface); 0 (Tetanus Toxin);24305-27-9 (Protirelin)

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Demonstration and characterization of partial glyceride specific lipases in pig thyroid plasma membranes. Nov 28 1980

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Defective regulation of the immune response to tetanus toxoid in Hashimoto's disease. Jul 1984

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Action of phospholipase C on the thyroid. Abolition of the response to thyroid-stimulating hormone.
Macchia V; Pastan I

Descriptors: Phospholipases--pharmacology--PD; * Thyroid Gland --drug [redacted]DE; *Thyrotropin--pharmacology--PD; Acetylcholine--pharmacology--PD; Clostridium --enzym[redacted]EN; Dogs; Edetic Acid--pharmacology--PD;Glucose--metabolism--ME; Neuraminidase--metabolism--ME
CAS Registry No.: 50-99-7 (Glucose); 51-84-3 (Acetylcholine); 60-00-4 (Edetic Acid); 9002-71-5 (Thyrotropin) Enzyme No.: EC 3.1.- (Phospholipases); EC 3.2.1.18 (Neuraminidase) Record Date Created: 19670706

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Beta-bu ngarotoxin-mediated liposome fusion: spectroscopic characterization by fluorescence and ESR. Oct 16 1990

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Snake-venom phospholipase A2 neurotoxins. Potentiation of a single-chain neurotoxin by the chaperon subunit of a two-component neurotoxin. Jan 15 1993

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Isolation and physiological characterization of taicatoxin, a complex toxin with specific effects on calcium channels. Nov 1992

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In[illegible] of phosphorylation of synapsin I and other synaptosomal pr[illegible]by beta-bungarotoxin, a phospholipase A2 neurotoxin. Dec 1992

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The complete sequence of the acidic subunit from Mojave toxin determined by Edman degradation and mass spectrometry. Mar 1 1990

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The effect of crotoxin on the release of acetylcholine and lactate dehydrogenase from rat brain cortical slices. 1992

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Specificity of action of beta-bungarotoxin on acetylcholine release from synaptosomes. May-Jun 1992

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Characterization and amino acid sequences of two lethal peptides isolated from venom of Wagler's pit viper, Trimeresurus wagleri. 1991

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Mode of action of alpha-latrotoxin: role of divalent cations in Ca2(+)-dependent and Ca2(+)-independent effects mediated by the toxin. Dec 1990

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Sequence homology between phospholipase and its inhibitor in snake venom. The primary structure of phospholipase A2 of vipoxin from the venom of the Bulgarian viper (Vipera ammodytes ammodytes, Serpentes). Apr 1987

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Binding of crotoxin, a presynaptic phospholipase A2 neurotoxin, to negatively charged phospholipid vesicles. Oct 1989

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The interaction between the presynaptic phospholipase neurotoxins beta-bungarotoxin and crotoxin and mixed detergent-phosphatidylcholine micelles. A comparison with non-neurotoxic snake venom phospholipases A2. Jul 5 1987

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Uncoupling of cholinergic synaptic vesicles by the presynaptic toxin beta-bungarotoxin. Oct 1986

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Neutralization of lethal potency and inhibition of enzymatic activity of a phospholipase A2 neurotoxin , crotoxin, by non-precipitating antibodies (Fab). Feb 13 1989

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[Effect o f melittin on ion currents in heart cell membranes] Deistvie melittina na ionnye toki membran kletok serdtsa. Jul-Aug 1987

11/6/94 05524017 92206154 PMID: 2562459
Crotoxi n, half-century of investigations on a phospholipase A2 neurotoxin. 1989

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Amino acid sequence of a presynaptic neurotoxin, agkistrodotoxin, from the venom of Agkistrodon halys Pallas. Feb 1989

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Binding of divalent and trivalent cations with crotoxin and with its phospholipase and its non-catalytic subunits: effects on enzymatic activity and on the interaction of phospholipase component with phospholipids. Nov 28 1989

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Preparation and characterization of monoclonal antibodies against beta-bungarotoxin and its A- and B-chains. 1989

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Ne[illegible]cular effects of a toxic phospholipase A2 and its nontoxic homologue from the venom of the sea snake, Laticauda colubrina. 1989

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[Amino acid sequence of orientotoxins I and II from the venom of the hornet Vespa orientalis] Aminokislotnaia posledovatel'nost' orientotoksinov I i II iz iada shershnia Vespa orientalis. Jan 1989

11/6/101 05187376 88163660 PMID: 3349062
Crotoxin, a phospholipase A2 neurotoxin from the South American rattlesnake Crotalus durissus terrificus: purification of several isoforms and comparison of their molecular structure and of their biological activities. Jan 26 1988

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Phospholipase A2 hydrolysis of membrane phospholipids causes structural alteration of the nicotinic acetylcholine receptor. Feb 8 1988

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Studies on the subunit structure of textilotoxin, a potent neurotoxin from the venom of the Australian common brown snake (Pseudonaja textilis). Sep 24 1987

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Binding ability of Clostridium botulinum neurotoxin to the synaptosome upon treatment of various kinds of the enzymes. Mar 30 1987

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Rattlesnake presynaptic neurotoxins: primary structure and evolutionary origin of the acidic subunit. Dec 3 1985

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Antibodies to beta-bungarotoxin and its phospholipase inactive derivative. Jun 22 1982

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Characterization of monoclonal antibodies against beta-bungarotoxin and their use as structural probes for related phospholipase A2 enzymes and presynaptic phospholipase neurotoxins. Jul 2 1984

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Sequence homology between phospholipase and its inhibitor in snake venom. The primary structure of the inhibitor of vipoxin from the venom of the Bulgarian viper (Vipera ammodytes ammodytes, Serpentes). Aug 1984

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Ceruleotoxin: identification in the venom of Bungarus fasciatus, molecular properties and importance of phospholipase A2 activity for neurotoxicity. 1983

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A comparison of nerve transection and chronic application of beta-bungarotoxin on acetylcholine receptor distribution and other nerve-muscle properties. Mar 1983

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Amino-acid sequence of the alpha-subunit of taipoxin, an extremely potent presynaptic neurotoxin from the Australian snake taipan (Oxyuranus s. scutellatus). Jun 1982

11/6/112 04337331 83079311 PMID: 7173209
Preparation of neurotoxic 3H-beta-bungarotoxin: demonstration of saturable binding to brain synapses and its inhibition by toxin I. Nov 1982

11/6/113 04282438 82200155 PMID: 7080027
Isolation of "ceruleotoxin" from Bungarus fasciatus venoms. 1982

11/6/114 04282437 82200154 PMID: 7080026
Synergism of the two subunits of crotoxin. 1982

11/6/115 04202883 81168081 PMID: 7217037
Amino acid sequence of phospholipase A from Bungarus multicinctus venom. Jan 1981

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Beta-bungarotoxin-induced cell-death of neurons in chick retina. Nov 4 1982

11/6/117 03822457 84104993 PMID: 6661452
[Release of 14C-acetylcholine from synaptosomes as affected by presynaptic neurotoxins from bee and cobra venoms--phospholipases A2] Vysvobozhdenie [14C] atsetilkholina iz sinaptosom pod vliianiem presinapticheskikh neirotoksinov iz iadov zmei i pchely--fosfolipaz A2. Nov 1983

11/6/118 03560161 80175361 PMID: 7370229
Phospholipase A2 activity and substrate specificity of snake venom presynaptic toxins. Mar 18 1980

11/6/119 03545041 80109855 PMID: 7353053
Penetrab ility of proteins through the digestive system of Sarcophaga falculata blowfly. Jan 3 1980

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beta-Bungarotoxin, a pre-synaptic toxin with enzymatic activity. Jan 1976

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Neuronal degeneration induced by stereotaxic injection of beta-bungarotoxin into rat brain. Feb 1979

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Relationship between the neurotoxicity and phospholipase A activity of beta-bungarotoxin. Jan 11 1977

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Saturable binding to cell membranes of the presynaptic neurotoxin, beta-bungarotoxin. May 21 1976

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The role of phospholipase activity in the action of a presynaptic neurotoxin from the venom of Notechis scutatus scutatus (Australian tiger snake). Jan 1 1976

11/6/125 02826941 76005594 PMID: 1158892
Amino acid sequence of a presynaptic neurotoxin from the venom of Notechis scutatus scutatus (Australian tiger snake). Sep 10 1975

11/6/126 02758131 77138542 PMID: 191284
Snake venom action: are enzymes involved in it? Feb 15 1977

11/6/127 02648802 80043375 PMID: 387107
Effect of presynaptic neurotoxin notechis II-5 from tiger snake venom on the motor nerve endings of mice] Deistvie presinapticheskogo neiroktoksina notekhisa II-5 iz iada tigrovoi zmei na dvigatel'nye nervnye okonchaniia myshi. Oct 1979

11/6/128 02559054 80046716 PMID: 499210
Postsynaptic effects of crotoxin and of its isolated subunits. Sep 1979

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Amino acid sequence of a postsynaptic neurotoxin from the venom of the Australian tiger snake Notechis scutatus scutatus. 1979

11/6/130 02481705 78053232 PMID: 928454
Selective enzymatic hydrolysis of nerve terminal phospholipids by beta-bungarotoxin: biochemical and morphological studies. 1977

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Central neurotoxicity of cobra neurotoxin, cardiotoxin and phospholipase A2. 1977

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Isolation and amino acid sequence of a neurotoxic phospholipase A from the venom of the Australian tiger snake Notechis scutatus scutatus. Dec 10 1976

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Are the blockade of nerve conduction and depolarization of skeletal muscle induced by cobra venom due to phospholipase A, neurotoxin or cardiotoxin? Jun 28 1972

11/7/46 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
08140009 94223544 PMID: 8169833
Inhibition of vacuolar adenosine triphosphatase antagonizes the effects of clostridial neurotoxins but not phospholipase A2 neurotoxins.
Simpson LL; Coffield JA; Bakry N
Department of Medicine, Jefferson Medical College, Philadelphia, Pennsylvania.
Journal of pharmacology and experimental therapeutics (UNITED STATES) Apr 1994, 269 (1) p256-62, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: NS-22153, NS, NINDS
Languages: ENGLISH Document type: Journal Article Record type: Completed
Bafilomycin A1, an inhibitor of vacuolar adenosine triphosphatase, was tested for its ability to antagonize botulinum neurotoxins (serotypes A-G), tetanus toxin and phospholipase A2 neurotoxins (notexin, beta-bungarotoxin, taipoxin and textilotoxin) on the mouse phrenic nerve-hemidiaphragm preparation. Bafilomycin itself produced concentration-dependent blockade of neuromuscular transmission without blocking nerve action potentials or muscle action potentials. This effect may have been due to inhibition of the proton pump that regulates acetylcholine transport into vesicles. At submaximal concentrations, bafilomycin was very effective in delaying the onset of paralysis due to all clostridial neurotoxins, but it had no protective effect against phospholipase A2 neurotoxins. Experiments were done to determine which of the three steps in clostridial neurotoxin action was antagonized by bafilomycin (e.g., binding, internalization and intracellular poisoning). Both pharmacological experiments and ligand-binding experiments showed that the drug did not block toxin binding to the plasma membrane. Similarly, pharmacological experiments on the time-dependent effects of bafilomycin showed that the drug did not antagonize the intracellular actions of toxins. The data indicated that bafilomycin acted at the intermediate step of internalization. This is in keeping with the facts that: 1) bafilomycin inhibits vacuolar adenosine triphosphatase, which in turn leads to inhibition of acidification in endosomes and 2) clostridial neurotoxins depend upon acidification of endosomes for translocation to the cytosol. The finding that bafilomycin antagonizes tetanus toxin may provide important clues for understanding how this toxin can act locally to produce flaccid paralysis. The finding that bafilomycin is a universal antagonist that protects against all clostridial neurotoxins may have important implications for developing therapeutic drugs. Record Date Created: 19940602

11/5/49 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
08081260 94066007 PMID: 8246147
Chelation of zinc antagonizes the neuromuscular blocking properties of the seven serotypes of botulinum neurotoxin as well as tetanus toxin.
Simpson LL; Coffield JA; Bakry N
Department of Medicine, Jefferson Medical College, Philadelphia, Pennsylvania.
Journal of pharmacology and experimental therapeutics (UNITED STATES) Nov 1993, 267 (2) p720-7, ISSN 0022-3565 Journal Code: JP3 Contract/Grant No.: NINCDS NS-22153, NS, NINDS Languages: ENGLISH Document type: Journal Article Record type: Completed
Botulinum neurotoxin types A, B (unactivated and activated), C, D, E, F and G, as well as tetanus toxin, paralyzed transmission in mouse phrenic nerve-hemidiaphragm preparations. Toxin-induced blockade of transmission was antagonized by chelators [e.g., ethylenediamine tetraacetic acid, tetrakis(2-pyridylmethyl)ethylenediamine or diethylene-triaminepentaacetic anhydride], but this effect was dependent on incubation conditions. Pretreatment of toxin with chelators failed to produce antagonism, but pretreatment of tissues did produce antagonism. Of the various chelators tested, tetrakis(2-pyridylmethyl)ethylenediamine produced the greatest effect. Antagonism of toxin-induced neuromuscular blockade could be partially reversed by washing chelators from tissues and could be fully reversed by adding an excess of zinc. The ability of chelators to antagonize clostridial neurotoxins was specific and did not extend to phospholipase A2 neurotoxins. Ligand-binding studies with radioiodinated toxin and brain membrane preparations showed that chelators did not antagonize toxicity by inhibiting toxin association with receptors. Similarly, pharmacological experiments with unlabeled toxin- and type-specific antibodies demonstrated that chelators did not act by blocking receptor-mediated internalization of toxin. The chelators appeared to exert their effects by antagonizing the intracellular actions of clostridial neurotoxins. Electrophysiological studies showed that chelators, at concentrations relevant to antagonism of botulinum neurotoxin and tetanus toxin, did not enhance transmitter release.(ABSTRACT TRUNCATED AT 250 WORDS) Record Date Created: 19940106
Tags: Animal; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Descriptors: *Botulinum Toxins--antagonists and inhibitors--AI;*Chelating Agents--pharmacology--PD; *Neuromuscular Blocking Agents--antagonists and inhibitors--AI; *Tetanus Toxin--antagonists and inhibitors--AI; *Zinc--metabolism--ME; Botulinum Toxins--classification--CL; Botulinum Toxins--metabolism--ME; Brain--metabolism--ME; Chelating Agents--metabolism--ME; Metalloendopeptidases--metabolism--ME; Mice;Neuromuscular Junction--drug effects--DE; Neuromuscular Junction--metabolism--ME; Rats; Sensitivity and Specificity; Serotyping; Tetanus Toxin--classification--CL; Tetanus Toxin--metabolism--ME
CAS Registry No.: 0 (Botulinum Toxins); 0 (Chelating Agents); 0 (Neuromuscular Blocking Agents); 0 (Tetanus Toxin); 7440-66-6 (Zinc)
Enzyme No.: EC 3.4.24 (Metalloendopeptidases)

11/7/104 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05067511 87184609 PMID: 3566763
Binding ability of Clostridium botulinum neurotoxin to the synaptosome upon treatment of various kinds of the enzymes.
Kitamura M; Sone S
Biochemical and biophysical research communications (UNITED STATES) Mar 30 1987, 143 (3) p928-33, ISSN 0006-291X Journal Code: 9Y8 Languages: ENGLISH Document type: Journal Article Record type: Completed
The binding ability of Cl. botulinum neurotoxin to synaptosomes upon treatment with various enzymes (neuraminidase, trypsin, and beta-bungarotoxin containing phospholipase A2 activity) was studied. When synaptosomes were treated with neuraminidase, their ability to bind toxin decreased; trypsin and beta-bungarotoxin had slightly week or no effect. The decrease in toxin-binding ability of synaptosomes was paralleled by a release of sialic acid from the synaptosomes by the neuraminidase treatment. The toxin-binding ability of synaptosomes treated with neuraminidase was lower than untreated ones at a high concentration of sodium chloride. The binding of the toxin to synaptosomes occurred at least at the two types of structural sites, one site which contained sialic acid, and other site which was sensitive to high ionic strength. It may be possible that another binding state except these is present at the synapse. Record Date Created: 19870518

17/6/1 10817918 20332947 PMID: 10874773
Ocular aspects of myasthenia gravis. 2000

17/6/2 10358931 20000569 PMID: 10532769
Strabismus surgery among aged medicare beneficiaries. Dec 1997

17/6/3 10240117 99371424 PMID: 10443824
The posterior thyroplasty window: anatomical considerations. Aug 1999

17/6/4 09825762 98378683 PMID: 9713061
Botulinum toxin A treatment of overactive corrugator supercilii in thyroid eye disease. May 1998

17/6/5 09428059 98018536 PMID: 9380360
Chemodenervation in treatment of upper eyelid retraction. 1997

17/6/6 08164191 94255188 PMID: 8196925
Control of eyelid retraction associated with Graves' disease with botulinum A toxin. Mar 1994

17/6/7 07433158 91214931 PMID: 1902375
Management of dysthyroid eye disease. Apr 1991

17/6/8 06895291 93064763 PMID: 1437204
Bilateral thyroarytenoid denervation: a new treatment for laryngeal hyperadduction disorders studied in the canine. Nov 1992

17/6/9 06668510 91032399 PMID: 2226978
Thyroid eye disease. 1990

17/6/10 06668494 91032402 PMID: 2226981
Botulinum toxin therapy in dysthyroid strabismus. 1990

17/6/11 06275988 87108295 PMID: 3804629
Botulinum in the treatment of adult motility disorders. Winter 1986

17/6/12 06220002 86204801 PMID: 3703521
Botulinum toxin for the treatment of dysthyroid ocular myopathy. Apr 1986

17/6/13 06008165 86099357 PMID: 3002078
[Myasthenic syndromes linked to mediator secretion disorders] Miastenicheskie sindromy, sviazannye s narusheniem sekretsii mediatora. 1985

17/6/14 05312485 89385497 PMID: 2779991
Botulinum toxin therapy of eye muscle disorders. Safety and effectiveness. American Academy of Ophthalmology. Sep 1989

17/6/15 05204825 89026356 PMID: 3179055
Botulinum toxin. Aug 1988

17/6/16 05131078 87070940 PMID: 3466462
Diplopia in thyroid eye disease. 1986

17/6/17 04905297 84225515 PMID: 6676980
Saccadic velocity measurements in strabismus. 1983

17/6/18 04576471 85026678 PMID: 6489104
Injection treatment of endocrine orbital myopathy. Aug 15 1984

17/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.

10240117 99371424 PMID: 10443824
The posterior thyroplasty window: anatomical considerations.
Maragos NE
Mayo Clinic, Rochester, Minnesota 55905, USA.
Laryngoscope (UNITED STATES) Aug 1999, 109 (8) p1228-31, ISSN 0023-852X Journal Code: L1W Languages: ENGLISH Document type: Journal Article Record type: Completed
OBJECTIVES: Explain surgical technique of performing a posterior thyroplasty window. Describe the internal laryngeal anatomy and structures available through the posterior window approach. Describe posterior window approach. STUDY DESIGN: Review of lateral laryngeal anatomy and retrospective review of 125 cases involving a posterior thyroplasty window approach. Review mechanics of stress and stress concentration inherent with partial removal of rigid substance. Describe anatomical considerations and surgical complications. METHODS: Charts were reviewed and tabulated for surgical complications, efficacy and safety of surgical approach, specific anatomical variations, and variety of surgery available through the posterior window. RESULTS: Performance of 125 posterior thyroplasty windows revealed no evidence of entry into the piriform sinus. Three thyroid ala fractures ensued, two of the body and one of the inferior cornu. Operations available included arytenoid adduction, arytenoid fixation, lysis of joint adhesions, and access to the posterior cricoarytenoid muscle for botulinum toxin injections. CONCLUSIONS: The posterior thyroplasty window affords easy, direct access to the internal, posterolateral larynx while preserving the cricothyroid joint, the action of the cricothyroid muscle, and the internal division of the recurrent laryngeal nerve. Record Date Created: 19990907

17/7/8 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
06895291 93064763 PMID: 1437204
Bilateral thyroarytenoid denervation: a new treatment for laryngeal hyperadduction disorders studied in the canine.
Sercarz JA; Berke GS; Ming Y; Rothschiller J; Graves MC
UCLA School of Medicine, Division of Head and Neck Surgery.
Otolaryngology--head and neck surgery (UNITED STATES) Nov 1992, 107 (5) p657-68, ISSN 0194-5998 Journal Code: ON8 Languages: ENGLISH Document type: Journal Article Record type: Completed
Adductor spasmodic dysphonia is a vocal disorder of uncertain etiology with no satisfactory long-term treatment. Recently, injection of botulinum toxin (Botax) into the thyroarytenoid (TA) muscle has been used as an effective temporary treatment. A surgical counterpart to bilateral TA Botox injection is described in this article. Bilateral thyroarytenoid denervation was performed through a window in the thyroid cartilage in seven canines, including four that were studied 3 months after the procedure. No serious complications occurred in the animals, each maintaining full vocal fold abduction and adduction. In all cases, anticipated physiologic changes in laryngeal function were observed, including the inability to generate high subglottic pressures during high levels of laryngeal nerve (RLN) stimulation. In two of the surviving animals, the ansa cervicalis was used to reinnervate the TA muscle, thereby preventing the possibility of reinnervation from the proximal RLN stump while limiting TA atrophy and fibrosis. Bilateral TA denervation represents a hopeful new long-term approach to spasmodic dysphonia treatment. Record Date Created: 19921218

17/7/15 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05204825 89026356 PMID: 3179055
Botulinum toxin.
Kowal L
Australian and New Zealand journal of ophthalmology (AUSTRALIA) Aug 1988, 16 (3) p264-6, ISSN 0814-9763 Journal Code: ANZ Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19881220

17/7/16 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
05131078 87070940 PMID: 3466462
Diplopia in thyroid eye disease.
Fells P; McCarry B
Transactions of the ophthalmological societies of the United Kingdom (ENGLAND) 1986, 105 (Pt 4) p413-23, ISSN 0078-5334 Journal Code: WA1 Languages: ENGLISH Document type: Journal Article Record type: Completed Record Date Created: 19870122

02jul01 08:32:48 User208600 Session D1405.1
File 155:MEDLINE(R) 1966-2001/Jul W2 (c) format only 2001
Dialog Corporation

Set	Items	Description
S1	6430	BOTUL?
S2	17401	CALCITONIN
S3	8	S1 AND S2
S4	19552	HYPOCALC? OR HYPERCALC?
S5	5	S1 AND S4
S6	90754	THYROID
S7	1231	S4 AND S6
S8	0	S1 AND S7
S9	23407	NEUROTOX?
S10	0	S7 AND S9
S11	1540602	TREAT?
S12	321	S11 AND S7

3/6/1 09948794 98433827 PMID: 9762863
Effects of botulinum neurotoxin type A on the expression of gephyrin in cat abducens motoneurons. Oct 12 1998

3/6/2 09471682 98014520 PMID: 9344569
Sensory and motor denervation influence epidermal thickness in rat foot glabrous skin. Oct 1997

3/6/3 09404508 97291189 PMID: 9145803
Expression of neurotransmitter genes in rat spinal motoneurons after chemodenervation with botulinum toxin. May 1997

3/6/4 09159891 97110858 PMID: 9081635
Regulation of motoneuronal calcitonin gene-related peptide (CGRP) during axonal growth and neuromuscular synaptic plasticity induced by botulinum toxin in rats. Apr 1996

3/6/5 09088502 96364144 PMID: 8739382
Upregulation of calcitonin gene-related peptide at mouse motor nerve terminals poisoned with botulinum type-A toxin. 1996

3/6/6 08461157 95123477 PMID: 7823160
Calcitonin gene-related peptide: possible role in formation and maintenance of neuromuscular junctions. Jan 1995

3/6/7 08356509 95187010 PMID: 7881294
Calcitonin gene-related peptide-like immunoreactivity, in botulinum toxin-paralysed rat muscles. Sep-Nov 1994

3/6/8 07960661 94061378 PMID: 8242236
Enhancement by calcitonin gene-related peptide of nicotinic receptor-operated noncontractile Ca2+ mobilization at the mouse neuromuscular junction. Oct 1993

3/7/5 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
09088502 96364144 PMID: 8739382
Upregulation of calcitonin gene-related peptide at mouse motor nerve terminals poisoned with botulinum type-A toxin.
Meunier FA; Colasante C; Faille L; Gastard M; Molgo J
Laboratoire de Neurobiologie Cellulaire et Moleculaire, C.N.R.S. (U.P.R.9040), Gif-sur-Yvette, France.
Pflugers Archiv (GERMANY) 1996, 431 (6 Suppl 2) pR297-8, ISSN 0031-6768 Journal Code: OZX
Languages: ENGLISH Document type: Journal Article Record type: Completed
Calcitonin gene-related peptide (CGRP)-like immunoreactivity of motor nerve terminals was investigated at different times after local in vivo injection of botulinum type-A toxin (BoNT/A) close to the mouse levator auris longus muscle. CGRP expression in most of control nerve terminals was undetectable, but markedly increased during muscle paralysis and synaptic remodelling and, declined once functional recovery occurred. Record Date Created: 19970116

5/7/1 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
10922488 20471417 PMID: 11021434
Fatal Clostridium botulinum toxicosis in eleven Holstein cattle fed round bale barley haylage.
Kelch WJ; Kerr LA; Pringle JK; Rohrbach BW; Whitlock RH
Department of Comparative Medicine, College of Veterinary Medicine, University of Tennessee, Knoxville 37901-1071, USA.
Journal of veterinary diagnostic investigation (UNITED STATES) Sep 2000 , 12 (5) p453-5, ISSN 1040-6387 Journal Code: A2D Languages: ENGLISH Document type: Journal Article Record type: Completed
Twenty-two lactating Holstein cattle in Tennessee had clinical signs of intoxication with preformed Clostridium botulinum toxin. These signs included weakness, paralysis of the tongue and chest muscles, abdominal breathing, and, in 11 of the 22 cows, death. Differential diagnoses included hypocalcemia , hypomagnesemia, carbohydrate overload, and several toxicoses including mycotoxin, lead, nitrate, organophosphate, atropine or atropine-like alkaloid, and botulism . A diagnosis of botulism by the ingestion of preformed C. botulinum type B toxin was made by eliminating these other diseases, by finding C. botulinum type B spores in 3 bales of round bale barley haylage fed to these cattle, and by isolating preformed type B toxin from 1 of the 3 bales. Confirmation of the toxin type was made by demonstrating mouse lethality by intraperitoneal injection of specimen extracts with neutralization by C. botulinum type B antitoxin. The haylage, harvested green and encased in black plastic bags to facilitate fermentation, was presumably contaminated by the botulinum toxin when fermentation failed to produce enough acid to lower the pH to 4.5, the pH below which C. botulinum growth is inhibited. Farmers and ranchers who use round hay balers to produce haylage should be alert to this potential problem. Record Date Created: 20010108

5/7/2 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
08679305 96124965 PMID: 8526819
The clinical differentiation of nervous and muscular locomotor disorders of sheep in Australia.
Bourke CA
NSW Agriculture, Agricultural Research and Veterinary Centre.
Australian veterinary journal (AUSTRALIA) Jun 1995, 72 (6) p228-34, ISSN 0005-0423 Journal Code: 9IE
Languages: ENGLISH Document type: Journal Article; Review; Review, Tutorial Record type: Completed
Many of the nervous and muscular locomotor disorders that affect sheep throughout Australia are commonly referred to as "staggers" syndromes. The range of clinical signs displayed by sheep suffering these disorders is sufficiently diverse to enable each syndrome to be graded into one of 5 progressive clinical groups. The first group, the limb paresis syndromes, includes the primary myopathies associated with the ingestion of Ixiolaena brevicompta, Malva parviflora, and Trachymene ochracea, as well as selenium and Vitamin E disorders, Paroo virus staggers, congenital progressive muscular dystrophy, humpy back, hypocalcaemic muscle weakness, Tribulus terrestris staggers and tetanus. The second group is characterised by limb paresis with knuckling of the fetlocks, and includes the plant-associated toxicities of Romulea rosea, Stachys arvensis, Trachyandra divaricata, and Tribulus micrococcus, together with haloxon toxicity, enzootic ataxia (copper deficiency), and the probably genetic disorders of segmental axonopathy, neuroaxonal dystrophy, and degenerative thoracic myelopathy. Other locomotor disorders that fit more loosely into this group are listerial myelitis (post-dipping staggers), vitamin A deficiency, cervico-thoracic vertebral subluxation Stypandra glauca toxicity, Ipomoea spp toxicity, ivermectin toxicity, and botulism . The third group, the falling syndromes, includes the probably genetic disorders of thalamic cerebellar neuropathy, cerebellar abiotrophy, and globoid cell leucodystrophy, together with Swainsona spp toxicity. The fourth group, the falling syndromes, includes the plant associated toxicities of phalaris staggers, perennial rye grass staggers and nervous ergotism (Claviceps paspali).(ABSTRACT TRUNCATED AT 250 WORDS) (40 Refs.) Record Date Created: 19960122

5/7/3 DIALOG(R)File 155:MEDLINE(R) (c) format only 2001 Dialog Corporation. All rts. reserv.
07689557 93149376 PMID: 1491765
[Primary hyperparathyroidism with prevalent neuro-muscular manifestations] Iperparatiroidismo primario con prevalenti manifestazioni neuro-muscolari.
Bartolucci L; Fioretti M; Fioroni E; Proietti MG; Gradoli C; Valori C
Istituto di Patologia Speciale Medica, Universita degli Studi di Perugia, Ospedale Civile, Terni.
Minerva medica (ITALY) Dec 1992, 83 (12) p841-5, ISSN 0026-4806 Journal Code: N6M
Languages: ITALIAN Document type: Journal Article Record type: Completed
A case of primary hyperparathyroidism with prevalent neuromuscular symptoms is described. Clinical, diagnostic and therapeutic implications are emphasized. Particular attention must involve a full clinical examination, electromyographic da neuromuscular biopsy to make differentiation from primary myopathy or denervation pathology. Some similarity of electromyographic data with those observed in botulism and myastenia gravis should also be taken in mind. Hypercalcemia could play a pathological role in conditioning abnormalities of nervous impulse conduction at the level of neuromuscular junction. Another possible interference might be related to a direct effect of parathormone and hypophosphataemia on nervous impulse conduction. "Glandular hyperplasia", as observed in this case at istologic examination, rises some problems as far as the prognosis is concerned. Record Date Created: 19930304

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